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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/536,804	11/10/2005	Magali Williamson	BJS-620-373	4496

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EXAMINER	
REDDIG, PETER J	
ART UNIT	PAPER NUMBER
1642	
MAIL DATE	DELIVERY MODE
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/536,804	WILLIAMSON ET AL.	
	Examiner	Art Unit	
	Peter J. Reddig	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *11 July 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *76-106,109 and 111-114* is/are pending in the application.

4a) Of the above claim(s) *76-105 and 112-114* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *106,109 and 111* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *11 July 2008* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. The Amendment filed July 11, 2008 in response to the Office Action of April 11, 2008 is acknowledged and has been entered. Previously pending claims 107, 108, and 110 have been cancelled and claims 106 and 109 have been amended. Claims 106, 109 and 111 are currently being examined as drawn to the species mutation site 5653 of the plexinB1 coding sequence and the A5653G mutation.

2. The following rejections are being maintained:

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

3. Claim 109 remains and claims 106 and 111 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention essentially for the reasons set forth in the Office Action of April 11, 2008.

Examiner argued:

Claim 107 refers to one or more mutation in a region of the nucleic acid which encodes, the cytoplasmic domain of the plexinB1 polypeptide, Claim 108 refers to one or more mutations at site 5653 of the plexinB1 coding sequence and claim 109 refers to the mutation A5653G. However, given that there is no point of reference given as to where the cytoplasmic domain of plexinB1 begins or ends and there is no point of reference given as to where the mutations of claim 108 and 109 are located, such as a SEQ ID NO: for plexinB1, the claims are indefinite as it cannot be determined to where these mutations are located.

Applicants argue that the Section 112, second paragraph, rejection of claims 107-109 is obviated by the above amendments. Applicants argue that claim 106 has been amended to specify that the listed positions refer to positions in the sequence of AB0007867.1. The

applicants believe that the person of ordinary skill in the art will readily appreciate the metes and bounds of the claims and the location of the recited mutations in the plexinB1 sequence.

Applicants arguments have been considered, but have not been found persuasive because a search of the number AB0007867.1 does produce any matches in any known databases, thus there is still no point of reference for the claimed mutations in claim 106. Furthermore, if Applicants meant to use the number AB007867.1, as used in the specification, this would still be indefinite as the database to which this number is associated is not cited anywhere in the specification. Furthermore, the use of accession numbers does not satisfy the requirements of 35 USC 112, second paragraph because accession numbers and the sequences corresponding to accession numbers are not unique identifiers required for nucleic acid because they can be modified, changed, and/or updated, and thus the cited sequence may vary or change over time. Thus, identifying a molecule by accession number does not provide a reliable unique identifier. Amendment of the claims to include unique identifiers which unambiguously define the claimed AB0007867.1 plexinB1 nucleic, such as a SEQ ID NO: for a sequence in a sequence listing would help to obviate this rejection.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4. Claims 106, 109 and 111 remain rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement for the reasons set forth in section 9, pages 4-11 of the Office Action of April 11, 2008.

Examiner argued:

One cannot extrapolate the teachings of the specification to the enablement of the claims because one of skill in the art would not be predictably able use changes in either the wild type plexinB1 or the A5653G mutant to identify or obtain a putative anti-cancer agent. Although the A5653G mutation is found in primary and metastatic prostate tumors, this same mutant plexinB1 reduces the tumorigenicity of cells *in vivo*. Thus, it is not clear if this mutation is a positive or negative regulator of prostate tumor or any tumor formation as the mutation appears to be associated with both positive and negative regulation of tumor formation and one of skill in the art would not predictably know what change in expression of the A5653G mutant B1 nucleic acid would be important for affecting tumor formation and would not predictably be able to identify and/or obtain a compound as a putative anti-cancer agent based on change in expression of the A5653G mutant plexin B1. Thus, undue experimentation would be required for identifying and/or obtaining a putative anti-cancer agent by the claimed method.

Additionally, it is not predictable that determining an increase in the wild-type plexinB1 would lead to the identification of a putative anti-cancer agent. Although the specification teaches that the expression of the wild-type plexin B1 suppresses tumor formation, Mack and Gish (US Pat. App. Pub. 2004/0005563, June 17, 2002) teach that plexin B1 is upregulated in ovarian cancer, see Table 14A and para. 0348 of the published application and Vogelstein et al. (US Pat. App. Pub 2005/0047996, October 9, 2001) teach that plexin B1 is upregulated in colorectal cancer, see Table 1. Thus, given that plexin B1 is upregulated in ovarian and colorectal cancers, the determination of an increase in the wild-type plexin B1 by a test compound would not predictably identify a putative anti-cancer agent. Thus, undue experimentation would be required for identifying and/or obtaining a putative anti-cancer agent by the claimed method.

Furthermore, given that A5653G mutant plexin B1 has only be identified in prostate cancers, one of skill in the art would not predictable expect that agents that affect the expression of this mutant plexinB1 nucleic acid would be putative anti-cancer agents for any cancer because it is well known in the art that cancers are heterogenous in phenotype and genes expressed and cancer therapeutics are not predictably effective for all cancers.

In particular, cancers comprise a broad group of malignant neoplasms divided into two categories, carcinoma and sarcoma. The carcinomas originate in epithelial tissues while sarcomas develop from connective tissues, see Taber's Cyclopedic Medical Dictionary (1985, F.A. Davis Company, Philadelphia, p. 274). Given that not all cancers originate from the same tissue types, it is known that cancers originate from different tissue types have different structures as well as etiologies and would present differently. Thus, it would not be predictably expected that a nexus, for example drawn to a connection between the A5653G mutant plexin B1 and prostate cancer, would be established between two cancer types that arose from different tissue types. Further, it is well known that even two carcinomas that present on the same organ have significant differences in etiology and genetic constitution. For example, Busken, C et al, (Digestive Disease Week Abstracts and Itinerary Planner, 2003, abstract No:850), teach that there is a difference in COX-2 expression with respect to intensity, homogeneity, localization and prognostic significance between adenocarcinoma of the cardia and distal esophagus, suggesting that these two cancers have different etiology and genetic constitution (last five lines of the abstract). Additionally, Kaiser (Science, 2006, 313: 1370) teaches that in a genomic analysis of mutations in breast and colon cancers, it was found that the cancer genes differ

between each colon and breast cancers and each tumor had a different pattern of mutations. Kaiser teaches that the steps to cancer may be more complex than had been anticipated, see 3rd col. Furthermore, Krontiris and Capizzi (Internal Medicine, 4th Edition, Editor-in-chief Jay Stein, Elsevier Science, 1994 Chapters 71-72, pages 699-729) teach that the various types of cancers have different causative agents, involve different cellular mechanisms, and, consequently, differ in treatment protocols. Chemotherapeutic agents are frequently useful against a specific type of neoplasm and there are no drugs broadly effective against all forms of cancer, see Carter, S. K. et a1. Chemotherapy of Cancer; Second edition; John Wiley & Sons : New York, 1981; appendix C. Given the above, it is clear that it is not possible to predictably extrapolate any potential correlation between an A5653G mutant plexin B1 directed anti-cancer agents and prostate cancer sensitivity to such an agent in any tumor type based on the information in the specification and known in the art without undue experimentation.

Furthermore, one of skill in the art would not predictably expect that all of the broadly claimed mutants of plexinB1 to be associated with cancer and thus an effect on their expression would not predictably be useful for identifying a compound as a putative anti-cancer agent. It is noted that the specification teaches that a mutant plexinB1 nucleic acid may comprise a nucleotide sequence which has one or more mutations relative to the wild-type plexinB1 nucleotide sequence, as set out in AB007867. The mutations may be deletions, insertions or substitutions of one or more nucleotides see para. 0014 of the published application. Given the above and given that claims are drawn to contacting "a" plexin B1 nucleic acid, which reads on fragments, which comprises one or more mutations in a coding region of the nucleic acid, the broadest reasonable interpretation of the claims is that the claims are not limited to any specific plexinB1 mutants and the plexinB1 mutants can comprise nucleic acids that are completely distinct from plexinB1. Furthermore, given claims 108 and 109 are indefinite in lacking a point of reference, these claims are also not limited to a particular site of mutation within the coding region of the plexinB1 nucleic acid and the plexinB1 mutants can comprise nucleic acids that are completely distinct from plexinB1.

It would not be expected that such a diverse array of mutants of plexin B1 would predictably be associated with cancer given that even naturally occurring gene variants, such as splice variants, do predictably have the same expression pattern or encode proteins with the same function as the related variants.. In particular, Benedict et al (J. Exp. Medicine, 2001, 193(1) 89-99) specifically teach that two splice isoforms of terminal deoxynucleotide transferase (a long form and a short form) enter the nucleus but have different activity, the long form does not catalyze nontemplated nucleotide addition but rather modulates the activity of the short form (see abstract). Jiang et al (JBC, 2003, 278(7) 4763-4769) specifically teach that the type 3 Ca2+ release channel, RyR3 exhibits strikingly different pharmacologic and functional properties depending on the tissues in which it resides. Upon examination, seven tissue specific alternatively spliced variants of RyR3 were detected. One of the variants was unable to form a functional channel but was able to suppress the activity of a different release channel. The authors conclude that tissue-specific expression of RyR3 splice variants is likely to account for some of the pharmacologic and functional heterogeneities of RyR3 (see abstract). The abstract of Matsushita et al (FEBS Letters, 1999, Vol. 443, pp. 348-352) teaches that latrophilins exhibit alternative splicing resulting in latrophilin-1, which is present in brain and

endocrine cells, latrophilin-2, which is ubiquitous, and latrophilin-3 which is brain-specific. The abstract of Singh et al (Glycobiology, 2001, Vol. 11, pp. 587-592) teaches that the CD44 splice variant, CD44v, is the major PNA-binding glycoprotein in colon cancer cells in contrast to standard CD44. These references serve to demonstrate that one of skill in the art cannot anticipate the biological activity of the proteins encoded by the broadly claimed plexinB1 mutants or the tissue distribution of the claimed mutants based on the biological activity of the protein encoded by the wild-type or tissue distribution of the wild-type nucleic acid or other mutants of plexinB1. Thus, even if it were found that the examination of the expression of the A5653G plexin B1 mutant could be used as claimed, undue experimentation would be required to use the broadly claimed mutants or even other mutations at position 5653 for the identification of putative anti-cancer agents.

The specification provides insufficient guidance with regard to the issues set forth above and provides insufficient working examples which would provide guidance to one skilled in the art and insufficient evidence has been provided which would allow one of skill in the art to predict that the invention would function as claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

Applicants argue that the Examiner is understood to believe that, although the A5653G plexin B1 mutant is shown to be present in prostate tumours, this mutant plexin B1 is also shown to reduce tumorigenicity in vivo; that the ordinarily skilled person could not predictably know what change in the expression of the A5653G plexin B1 mutant would be important for affecting tumour formation and would not therefore be able to predictably identify and/or obtain an anti-cancer agent based on a change in expression of the A5653G plexin B1 mutant.

Applicants argue that, the transfected NIH3T3 cells used in the in vivo tumorigenicity experiments in fact produced little or no mutant or wild-type plexinB1 protein. The in vivo tumorigenicity experiments described in the specification therefore do not allow any conclusions to be drawn about the effect of wild type and mutant plexinB1 protein on tumourigenicity.

Applicants argue that however, the applicant submit that the data set out in the originally-filed application on pages 51 line 16 to page 52 line 14, page 53 line 4 to page 55 line 13 and page 56 line 10 to page 56 line 26 show that the A5653G and other mutations in the plexinB1

coding sequence present in a high proportion of tumour samples and not present in healthy tissue samples and therefore display a strong positive correlation with the formation of breast and prostate tumours.

Applicants argue that in the light of this strong positive correlation, the applicants believe that one of ordinary skill in the art would reasonably predict that a decrease in expression of the mutant plexin B1 would be important for reducing the formation of tumours and would therefore be able to predictably identify and/or obtain an anti-cancer compound based on a decrease in expression of the A5653G mutant and other mutant plexinB1 nucleic acids.

Applicants' arguments have been considered, but have not been found persuasive because the presence of the A5653G mutation in plexin B1 in prostate tumors does not indicate whether or not this mutation, or any other mutation, in plexin B1 is causative for prostate cancer or any other cancer. Thus, in the absence of the deleted *in vivo* mouse tumor model data, it can not be determined if this mutation or any other mutation in plexin B1 is important in the etiology of cancer. Although the A5653G mutation was shown to induce anchorage independent growth in NIH-3T3 cells (still in Fig. 2 of the amended specification), given that Applicants have stated on the record that the transfected NIH3T3 cells expressed little or no protein, and the experiments employing these cells provide no information about the effect of wild type or mutant plexin B1 on anchorage independent growth or tumorigenicity (see page 10 of the Remarks of 7/11/2008), one of skill in the art can not predictably use claimed method for the identification of an anticancer drug because the presence of a mutation in a cancer does not indicate that the mutation is involved in the etiology of cancer.

Applicants argue that the Examiner is further understood to believe that one could not predictably extrapolate from prostate cancer to any tumour type without undue experimentation.

Applicants argue that the applicants believe however that the data set out in the originally-filed application on pages 51 line 16 to page 52 line 14 and page 53 line 4 to page 55 line 13 show that the A5653G mutation and the other listed mutations in the plexinB1 coding sequence display a strong positive correlation with the formation of prostate tumours.

Applicants argue that the data set out in the originally-filed application on page 56 lines 10 to 26 show that the A5653G mutation and the other listed mutations in the plexinB1 coding sequence display a strong positive correlation with the formation of breast tumours.

Applicants argue that a person of ordinary skill in the art could predictably extrapolate the described correlation between mutant plexin B1 and tumour formation to breast and prostate tumours without undue experimentation based on the information in the specification and known in the art.

Applicants' arguments have been considered, but have not been found persuasive because the presence of the A5653G mutation in plexin B1 in prostate tumors does not indicate whether or not this mutation, or any other mutation, in plexin B1 is causative for prostate cancer or any other cancer in the absence of data showing that it is involved in the etiology of cancer formation as set forth above.

Applicants argue that the Examiner further understood to believe that that one of ordinary skill in the art would not predictably expect that all of the claimed mutants of plexin B1 are associated with cancer and thus an effect on their expression would allegedly not be predictably useful for identifying a compound as a putative anti-cancer agent.

The instant claims recite mutations located at specified positions in the coding sequence of plexinB1 which are identified by reference to the sequence of database and publicly available entry AB0007867.1. Mutations located at all of the specified positions are shown in the specification to correlate with prostate and/or breast cancer.

Applicants' arguments have been considered, but have not been found persuasive because the sequence of AB0007867.1 is not publicly available and thus it can not be determined whether or not the claimed mutations are the mutations identified in plexin B1 in the specification. Thus, it cannot predictably be determined if the claimed mutations are in any way associated with cancer.

Applicants argue that mutations at these positions alter an amino acid in the encoded plexinB1 protein. Many of these amino acids are at sites which are conserved in evolution and therefore are likely to be important functionally. Moreover, the applicants submit that all the mutants studied to date alter cell function. Applicants argue that the ordinarily skilled person would be able to reasonably extrapolate from the data in the application that any mutation at these positions which alters the encoded amino acid residue in the plexinB1 protein will have a similar effect. Applicants argue that one of ordinary skill in the art would therefore expect that all of the claimed mutants of plexin B1 were associated with cancer, in the light of the data set out in the specification, and thus an effect on the expression of these mutants would be predictably useful for identifying a compound as a putative anti-cancer agent.

Applicant's arguments have been considered, but have not been found persuasive because Applicants not provided any evidence that the mutations affect cell function are critical in the etiology of any cancer as Applicants have removed the functional data from the specification

from the specification and have stated, as set forth above, that the functional data presented with the NIH3T3 cells transfected with the plexin B1 mutants provide no information about the effect of the mutants on anchorage independent growth or tumorgenicity. Thus, whether or not the amino sites of the claimed mutations are conserved in evolution, one of skill in the art could not predictably use the claimed method for the identification of a putative anti-cancer agent in the absence of further guidance as to the functional importance of the mutants in the etiology of cancer.

Applicant's arguments have not been found persuasive and the rejection is maintained.

New Grounds of Rejection

Claim Rejections - 35 USC § 112

5. Claims 106, 109, and 111 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a new matter rejection.

The limitation of a "the plexinB1 coding sequence of AB0007867.1" claimed in Claims 106, 109, and 111 has no clear support in the specification and the claims as originally filed. A review of the specification as revealed support for AB007867.1, see page 8, line 29. Thus subject matter claimed in Claims 106, 109, and 111 broadens the scope of the invention as originally disclosed in the specification.

Drawings

6. The drawings were received on 7/11/2008. These drawings are accepted.

7. All other objections and rejections recited in Office Action of April 11, 2008 are withdrawn.

8. No claims allowed.

9. This action is a **final rejection** and is intended to close the prosecution of this application. Applicant's reply under 37 CFR 1.113 to this action is limited either to an appeal to the Board of Patent Appeals and Interferences or to an amendment complying with the requirements set forth below.

If applicant should desire to appeal any rejection made by the examiner, a Notice of Appeal must be filed within the period for reply identifying the rejected claim or claims appealed. The Notice of Appeal must be accompanied by the required appeal fee.

If applicant should desire to file an amendment, entry of a proposed amendment after final rejection cannot be made as a matter of right unless it merely cancels claims or complies with a formal requirement made earlier. Amendments touching the merits of the application which otherwise might not be proper may be admitted upon a showing a good and sufficient reasons why they are necessary and why they were not presented earlier.

A reply under 37 CFR 1.113 to a final rejection must include the appeal form, or cancellation of, each rejected claim. The filing of an amendment after final rejection, whether or not it is entered, does not stop the running of the statutory period for reply to the final rejection unless the examiner holds the claims to be in condition for allowance. Accordingly, if a Notice of Appeal has not been filed properly within the period for reply, or any extension of this period obtained under either 37 CFR 1.136(a) or (b), the application will become abandoned.

10. Applicant's amendment necessitated the new grounds of rejection. Thus, **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 C.F.R. 1.136(a).

A SHORTENED STATUTORY PERIOD FOR RESPONSE TO THIS FINAL ACTION IS SET TO EXPIRE THREE MONTHS FROM THE DATE OF THIS ACTION. IN THE EVENT A FIRST RESPONSE IS FILED WITHIN TWO MONTHS OF THE MAILING DATE OF THIS FINAL ACTION AND THE ADVISORY ACTION IS NOT MAILED UNTIL

AFTER THE END OF THE THREE-MONTH SHORTENED STATUTORY PERIOD, THEN THE SHORTENED STATUTORY PERIOD WILL EXPIRE ON THE DATE THE ADVISORY ACTION IS MAILED, AND ANY EXTENSION FEE PURSUANT TO 37 C.F.R. 1.136(a) WILL BE CALCULATED FROM THE MAILING DATE OF THE ADVISORY ACTION. IN NO EVENT WILL THE STATUTORY PERIOD FOR RESPONSE EXPIRE LATER THAN SIX MONTHS FROM THE DATE OF THIS FINAL ACTION.

11. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Peter J. Reddig whose telephone number is (571) 272-9031. The examiner can normally be reached on M-F 8:30 a.m.-5:00 p.m..

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms can be reached on (571) 272-0832. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Peter J Reddig/

Examiner, Art Unit 1642

/Karen A Canella/

Primary Examiner, Art Unit 1643